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NOTES ON THE RELATION BETWEEN COLIFORMS AND ENTERIC PATHOGENS [1] [2]

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In order that a clear perspective be maintained regarding the value of the coliform [3] test as an indicator of pollution and, therefore, of possible infection with enteric pathogens, it seems worthwhile to present evidence tending to clarify the relationship between coliforms and pathogens. This is particularly true at present inasmuch as the coliform test (*1*) has recently received an adverse decision by the Illinois Supreme Court as to its value in indicating an unsafe water.

During the period of establishment of the coliform group as an indicator of unsafe waters, considerable effort was devoted to the isolation of enteric pathogens, particularly *Eberthella typhosa*, and to the relative rates of decrease of coliforms and enteric pathogens under various conditions. Methods for quantitative isolation of such pathogens were, however, less effective than those available now. The introduction, in 1927, of Wilson and Blair's bismuth sulfite agar has resulted in much work being done on the isolation of certain enteric pathogens during the past 10 or 12 years by a number of

[1] From the Division of Public Health Methods, National Institute of Health, Stream Pollution Investigations, Cincinnati, Ohio.

[2] This paper consists, in part, of certain sections of an unpublished report entitled "A report on the public health aspects of clamming in Raritan Bay," by Robert W. Kehr, Benjamin S. Levine, Chester T. Butterfield, and Arthur P. Miller. The original report includes extensive laboratory studies of pollution of Raritan Bay made under supervision of the U. S. Public Health Service by the New York State Conservation Department, the New Jersey State Department of Health, and the New York City Department of Health.

[3] "Coliform" bacteria are considered as including all aerobic and facultative anaerobic Gram negative nonsporeforming bacteria which ferment lactose with gas formation. This group, as defined, is equivalent to (1) the "*B. coli*" group as used in all editions of Standard Methods of Water Analysis prior to the sixth, (2) the term "coli-aerogenes" group as used in the sixth, seventh, and eighth editions of Standard Methods of Water Analysis, and (3) the term "colon group" in Standard Methods of Milk Analysis.

*It is noted with deep regret that an airplane on which Mr. Kehr was traveling in connection with his work on the Alaska Highway Project disappeared on December 20, 1942, and that no trace of the missing plane had been discovered up to the present writing in March 1943.

investigators. These workers have been successful in isolating *E. typhosa* from sewage and polluted waters, using Wilson and Blair's media or various modifications of it. While it hardly seems necessary to review the literature extensively in this paper, several of these workers, or groups of workers, have made numerous isolations of *E. typhosa* and other enteric pathogens. The work of the London Metropolitan Water Board Laboratories is perhaps the most complete and carefully controlled. Inaugurated by the late Sir Alexander Houston, each report from 1927 to 1938 (*2* to *13*) carries some reference to isolations of enteric pathogens and the total amount of this work is very considerable. It includes isolations or attempted isolations from sewage, sewage treatment plant effluents, and raw Thames River water. A number of the experiments were controlled by adding a known concentration of *E. typhosa* to half of the sample and increasing the estimated concentration of typhosa present by the ratio of $\frac{\textit{E. typhosa} \text{ added to control}}{\textit{E. typhosa} \text{ recovered from control}}$. Such results also have value when isolations are not accomplished from the sample, inasmuch as this probably justifies the assumption that on the average *E. typhosa* was absent from a fraction of the original sample equal to the ratio $\frac{\textit{E. typhosa} \text{ recovered}}{\textit{E. typhosa} \text{ added}}$ in the control. During the years 1931–1938, the London Metropolitan Water Board Laboratories also examined samples of raw sewage and effluent from the Epping sewage treatment works for *Salmonella schottmuelleri* (*B. paratyphosus B.*). This organism was found to be present in large numbers following an outbreak of 260 cases of paratyphoid at Epping during 1931, a smaller outbreak of 22 cases in 1933, and 2 cases in 1935. This work, together with other studies of a more limited scope by the London Water Board group, constitutes the largest mass of data available on the isolation of enteric pathogens from water and sewage. Wilson (*14*, *15*, *16*) and Wilson and Blair (*17*) have reported numerous isolations of *E. typhosa* from polluted waters, sewage, and the shell liquor of cockles (*14*).

Green and Beard (*18*) have reported the isolation of *E. typhosa* from Palo Alto sewage in 9 of 55 1-ml. samples; Ruchhoft (*19*) has reported isolations in 2 0.1-ml. samples of Chicago activated sludge, while Heukelekian and Schulhoff (*20*) reported failure to isolate *E. typhosa* from the sewages of 15 municipalities in 0.1-ml. amounts. Stewart and Ghosal (*21*) reported isolation of *E. typhosa* from the River Hooghly in India. Hajna (*22*) has reported isolations of *E. typhosa* from 6 of 22 samples of crude sewage, none of the 9 samples of effluent, 3 of 7 samples of raw sludge, and none of 3 samples of digested sludge from Baltimore and vicinity. From this series of

samples 22 strains of *E. typhosa* were confirmed by Hajna. Two *E. typhosa* were isolated by Hajna from 1 ml. of raw sewage containing 11 billion [4] *Esch. coli* per ml.

Mom and Schaeffer (*23*) reported an extensive series of isolations from sewage, sludge, and river water at Bandoeng, Dutch East Indies, where the morbidity rates for typhoid fever are around 30 cases per thousand per year. Wilson (*14*) and Mom and Schaeffer (*23*) stress the relationship between the typhoid morbidity rate and the concentration of *E. typhosa* found in the sewage of a community. Accordingly, available data have been plotted (fig. 1) in order to give some idea of the relationship between reported isolations and the normal prevalence of typhoid fever in the community. The concentration of *E. typhosa* is expressed as *E. typhosa* per million coliforms, inasmuch

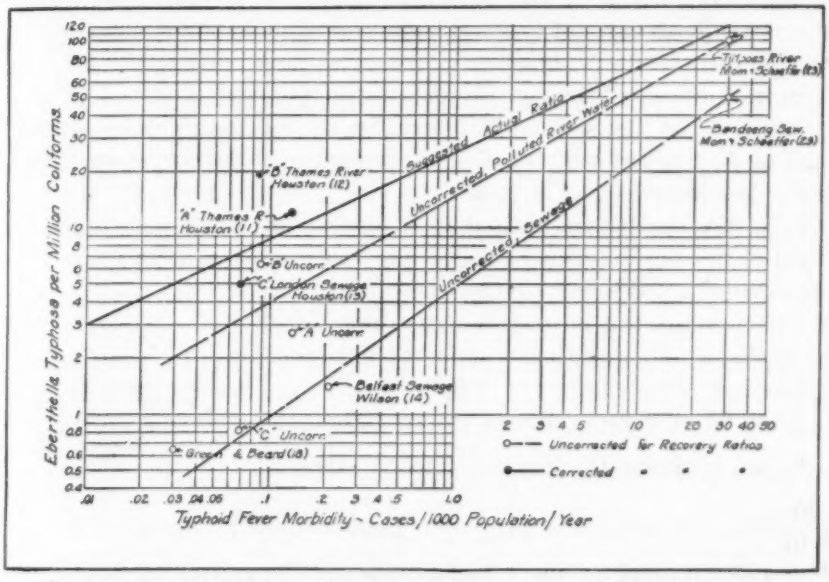

FIGURE 1.—*Eberthella typhosa* per million coliforms for varying typhoid fever morbidity rates.

as this gives an increasing value for increasing prevalence and is convenient to remember, being about the concentration per ml. in a relatively strong raw sewage.

The positions of the lines on figure 1 are rather crude as the data available are insufficient to determine the relationship with any degree of accuracy. The nature of the curve between 0.2 and 30 cases per 1,000 population per year is entirely unknown and the curves are plotted as linear on logarithmic paper without other than convenience for justification. There are also many factors such as individual technique of the investigators, variations in the composition of the Wilson and Blair agar, and the methods of determination and interpretation of the coliform group organisms. All of these factors influ-

[4] The 11 billion coli per ml. of sewage is rather high and may be a misprint in the original article.

ence the results as plotted and it has not been possible to take them into account. The upper line is the suggested ratio, as indicated by the data available, for the absolute numbers of *E. typhosa* in the absence of epidemic conditions, while the two lower lines indicate the ratios which might be expected, uncorrected for recovery losses, in polluted river waters and sewage. The methods used in all cases were direct plating on Wilson and Blair's agar (or some slight modification thereof) except for raw Thames River water where the bacteria were concentrated by precipitation with alum. The ratios given in figure 1 are the approximate minimum ratios to be expected in general in sewage and waters polluted by sewage from large or fairly large populations. These ratios would be increased by carelessness in disposal of infected excreta or the presence of epidemic conditions. In the absence of both cases of typhoid fever and typhoid carriers there would presumably be no *E. typhosa* in the sewage, a condition which may occur in some small towns. Similarly, much greater fluctuations might be expected in the sewage from smaller towns due to the large numbers of *E. typhosa* which a single carrier can excrete and the lack of the balancing effect of large populations.

It is interesting to note that a variety of pathogens have been isolated in examinations of water and sewage by some of the workers previously mentioned. *Salmonella schottmuelleri* (*B. paratyphosus B.*) is perhaps the most frequently mentioned pathogen other than *E. typhosa*. In addition to routine examination and isolation from the sewage and effluent of Epping during the years 1931–1938, the London Metropolitan Water Board Laboratories have reported: One isolation (*4*) from 24 samples of six sewages, mean $\frac{S.\ schottmuelleri}{\text{million coliforms}} = \frac{20}{2.67} = 7.5$; 3 isolations (*3*) from 1.825 ml. of 11 different sewages. The 1931 Report of the Chief Medical Officer, British Ministry of Health (*24*), mentions the isolation of *S. schottmuelleri* in 2-ml. amounts from the effluent at Wroxall and in 100-ml. amounts from the River Var 3½ miles below the point of discharge of Wroxall effluent. Gray (*25*) and Begbie and Gibson (*26*) have reported isolation of *S. schottmuelleri* from Edinburgh sewage.

Isolations of *Salmonella typhimurium* (*B. aertrycke*) have been reported by the Metropolitan Water Board (*3, 6*) and in the 1931 Report of the Chief Medical Officer, British Ministry of Health (*24*). The latter also reported the isolation of *Salmonella newport* (*B. newport*) from Ipswich sewage and *Salmonella enteritidis* (*B. enteritidis, Gaertner*) from Daventry sewage.

Most of the isolations reported for organisms other than *E. typhosa* and *S. schottmuelleri* (*B. paratyphosus B.*) were made incidental to the search for *E. typhosa*, and in view of the general lack of knowledge

of percentages of recovery little significance can be attached to the quantitative aspects of these reported isolations other than *E. typhosa* and to some extent *S. schottmuelleri.*

STABILITY OF THE *E. TYPHOSA*–COLIFORM RATIO

The expression of *E. typhosa* concentrations in both sewage and polluted waters as a ratio, such as the *E. typhosa* per million coliforms as used in figure 1, implies the existence of equal logarithmic rates of decrease with time of these organisms under the same environment. There is much to support such an assumption, however, in that many of the factors which combine to form "natural purification rates," such as ingestion by protozoa and sedimentation, would not be expected to differentiate between groups of organisms which do not differ too greatly in essential characteristics such as optimum growth requirements, size, and motility. Perhaps the best evidence of equal rates of decrease yet collected is furnished by the work of the London Metropolitan Water Board in isolations of *Salmonella schottmuelleri* (*B. paratyphosus B.*) from the sewage and effluent at Epping over a period of years. These results, usually 17 samples each of sewage and effluent per year, have been summarized in table 1, which presents the median ratio of $\frac{\text{coli}}{S.\ schottmuelleri}$ found in Epping sewage and effluent. It will be noted that the mean of the median ratios is 1518 for raw sewage as against 1105 for final effluent. This greater ratio in raw sewage is about what one would expect if *S. schottmuelleri* are more difficult to isolate from raw sewage than final effluent. Sir Alexander Houston was able to recover 1/3.1 (*12*) and 1/4.8 (*11*) of *E. typhosa* added to river water, but only 1/10.9 (*13*) of the same organism added to raw London (Barking) sewage.

TABLE 1.—*Ratios of coli*/S. schottmuelleri *in raw sewage and effluent from Epping, England*

Year	Reference	Median coli/*S. schottmuelleri*		Percent coli reductions	Method of treatment
		Sewage	Effluent		
1931	2	278	[1] 190	96.8	Land treatment.
1932	3	156	[2] >300	85.5	Do.
1933	4	992	250	94.1	Do.
1934	5	714	[2] >300	95.5	Do.
1935	6	5,000	1,500	98.7	Trickling filters.
1936	7	1,000	3,000	98.7	Do.
1937	8	2,000	[2] >300	99.2	Do.
1938	9	2,000	3,000	99.3	Do.
Mean		1,518	1,105	97.0	

[1] Omitting effluent samples prior to May 11 to secure comparable data.
[2] *Salmonella schottmuelleri* not isolated from quantities of effluent containing median numbers of coliforms. Median would be slightly higher if these results were omitted.

Considering the fact that each pair of these samples has undergone identical purification varying between 85.5 and 99.3 percent, such purification being essentially an accelerated natural purification, the net result constitutes the strongest sort of evidence justifying the use of enteric pathogen/coliform ratios for natural purification processes probably up to 99.9 percent reductions from raw or diluted raw sewage. Beyond that percent purification, it seems likely that additional evidence will be required as to relative reductions of pathogens and the coliform group as most curves for coliforms tend to slacken from a straight logarithmic rate of decrease, probably somewhat sooner than the rates of decrease for the more limited number of strains of enteric pathogens.

Additional, though somewhat indirect, supporting evidence for parallel reductions of pathogens and coliforms is presented by Ruchhoft (*27*) and his coworkers in their studies of decrease of coliform group organisms in the Illinois River. The authors conclude, "These results indicate that during self-purification of polluted water there is comparatively little change in the ratio of *Bact. coli* to *Bact. aerogenes*."

RATES OF DECREASE OF *E. TYPHOSA* AND COLIFORMS UNDER CONDITIONS OF NATURAL PURIFICATION

A large number of investigators have studied the rates of decrease of *E. typhosa* under a variety of conditions. These studies are rarely directly comparable, and frequently were not strictly quantitative. Many of the earlier studies were based on an absence of *E. typhosa* from a qualitative test only, for this organism; the results are therefore of limited value in quantitative studies. Much of the work has been carried on under conditions which were quite surely anaerobic and varied widely from normal, natural purification in streams and quiescent bodies of water.

In figures 2, 3, and 4 are shown data on rates of decrease of *E. typhosa* published by various investigators, compared to values for the rates of decrease of coliforms and 37° agar count organisms as found on the Scioto River (*28*).

The rates of decrease of coliforms in the Scioto River at low stages of the river were found to be about equal in the upper, heavily polluted section of the river and in the lower, less polluted section over concentration ranges that varied by as much as 99.9 percent or more. Observations on the Scioto and the data shown in figures 2, 3, and 4 are divided into three temperature ranges, 9.9° C. and below (fig. 2), 10–19.9° C. (fig. 3), and 20° C. and above (fig. 4), probably averaging, for natural waters, about 5° C., 15° C., and slightly under 25° C., respectively. Very little data are available for figure 2 but Heukelekian and Schulhoff (*20*) presented figures for Raritan River water at

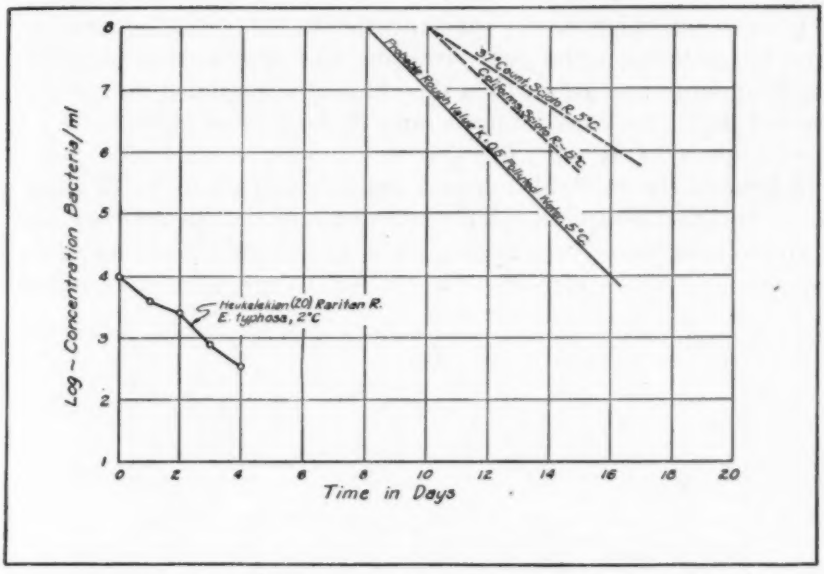

FIGURE 2.—Rates of decrease, *E. typhosa* and coliforms, 9.9°C. and below.

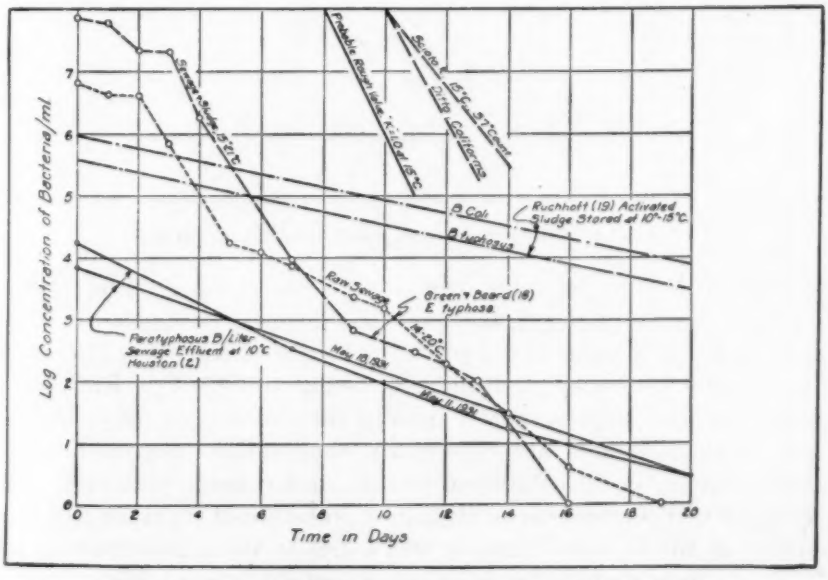

FIGURE 3.—Rates of decrease, *E. typhosa* and coliforms, 10° to 19.9°C.

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2° C. which were only slightly lower than comparable rates of coliform decrease in the Scioto River (*28*). If the usual form of monomolecular curve, $y=b10^{-Kt}$, is chosen as representing the logarithmic rate of decrease, where y equals the concentration of bacteria after time, t, in days, b equals the initial concentration, and K a constant dependent largely upon temperature, then the value of K assumed for a temperature of 5° C. for both coliforms and *E. typhosa* as indicated by the data in figure 2 is roughly 0.5.

In figure 3 the available evidence for determination of a K value at 15° C. is rather scattered. The data of Green and Beard (*18*), based on stored sewage and sewage plus sludge at 14°–20° C. and 15°–21° C., respectively, have a K value of about 0.8–1 during an early logarith-

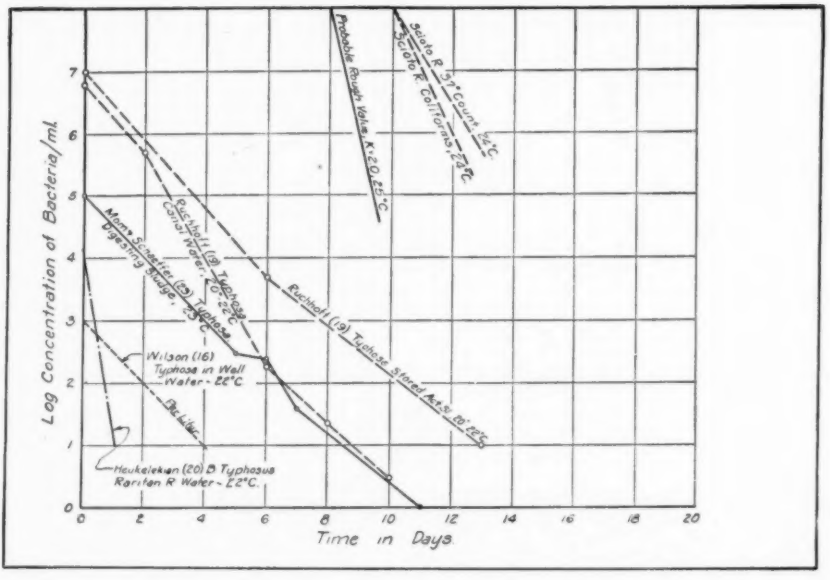

FIGURE 4.—Rates of decrease, *E. typhosa* and coliforms, 20° C. and above.

mic rate of decline, but this decreases to about 0.5 at later stages. Houston's figures (*2*) for *S. schottmuelleri* in sewage effluent at 10° C. show roughly a K value of 0.3 while the figures of Ruchhoft (*19*) show K values of 0.1 for activated sludge stored at 10°–15° C. Ruchhoft's figures also show approximately parallel rates of decline for coliforms. None of these figures are reasonably comparable to conditions in natural waters, being conducted mostly under anaerobic conditions. A K value of 1.0 would seem, therefore, to be about right for the temperature of 15° C., based largely on the Scioto River observations for coliforms, supported by the parallel rates of decrease of coliforms and *E. typhosa* under identical conditions as shown by Ruchhoft.

In figure 4, Heukelekian and Schulhoff (*20*) show K values of about 3.0 for Raritan River water at 22° C., Wilson (*16*) a K value of about 0.5 for well water at 22° C., Mom and Schaeffer (*23*) values of 0.5 for digesting sludge decreasing somewhat after about 99.5 percent drop in *E. typhosa*. Ruchhoft (*19*) gives rates of decrease of slightly under 1.0 for canal water at 20°–22° C. and slightly over 0.5 for stored

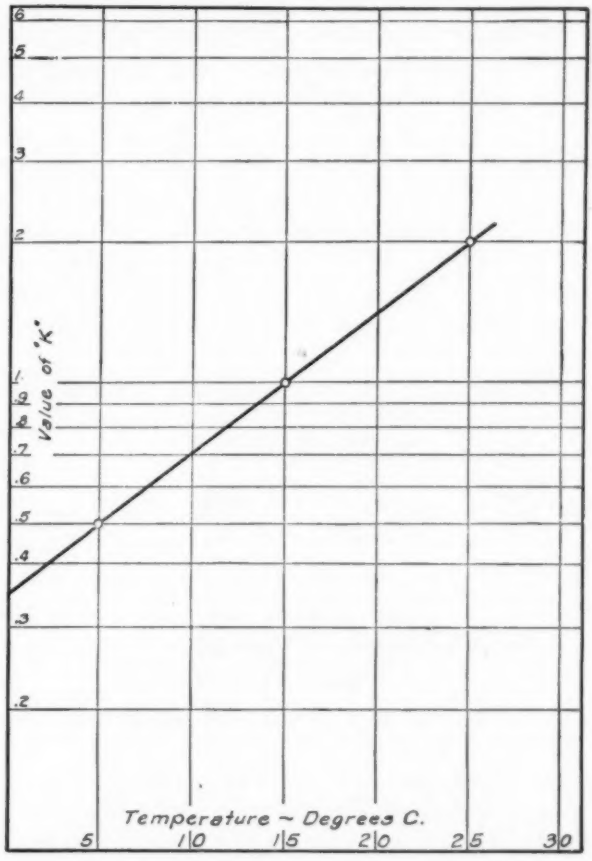

FIGURE 5.—Chart showing variation in value of "K" with temperature in degrees C.

activated sludge at 20°–22° C. A rough K value of 2.0 would probably represent the rate of decrease at 25° C. for both coliforms and *E. typhosa*, due to natural purification processes. The relationship thus obtained between the rates of decrease and temperature is shown, plotted on semi log paper, in figure 5.

If the rates of decrease of coliforms and *E. typhosa* in natural waters are equal for decreases up to 99.9 percent, then a constant typhosa/coliform ratio could be assumed for 1.5 days at 25° C., 3 days at 15° C., and 6 days at 5° C., starting with fresh pollution such as sewage or

dilute sewage and using the values of K derived from the data presented in figures 2, 3, and 4.

The assumption of typhosa/coliform ratios is a logical and rational approach to a more exact and quantitative use of the estimation of coliforms as an indicator of sanitary conditions. This ratio is supported by considerable evidence as presented above and should give additional, if not absolute, legal status to the coliform test which recently received an adverse decision in the courts of Illinois (*1*). This study presents little more than a start on the work needed before such a ratio could have much practical value, inasmuch as the limits of its usefulness should be clearly defined. Some of the factors which should be investigated are: (1) variations to be expected in the ratio in different sewages, (2) the changes, if any, encountered at low coliform concentrations due to high rates of natural or chemical (particularly chlorination) purification, (3) the significance of coliforms from sources other than sewage, and (4) whether, in the presence of coliforms largely from sources other than sewage, isolation of enteric pathogens can be made.

It is believed that the actual determination of the typhosa/coliform ratio and its variations would be worth while in many instances, particularly where a polluted water is used as a source of supply for domestic purposes.

A POSSIBLE THEORETICAL APPLICATION OF THE *E. TYPHOSA*/COLIFORM RATIO

Based on the *E. typhosa*/coliform ratios shown in figure 1, and recorded water-borne outbreaks of typhoid fever, it becomes possible to estimate quite roughly a theoretical minimum infectious dose of *E. typhosa* for the general population and the percentage of persons infected by that dosage.

It is desired to separate epidemics of typhoid fever more or less arbitrarily into two groups according to the presumed intensity of infection and to discuss at length only those of presumably light infectious dosages.

In the category of heavy or uncertain infectious dosages would be included most carrier-borne typhoid, most milk-borne typhoid, and water-borne typhoid with rather heavy attack rates for the population exposed or where the epidemic was traced rather definitely to more or less direct carrier or patient discharge. The assumption of a heavy attack rate accompanying ingestions of large numbers of *E. typhosa* is in accord with general experience in infections and the reverse would also hold true with reservations regarding the size of the minimum infectious dose.

An interesting small outbreak of typhoid fever, with presumably heavy infectious dosages, was reported by Morales and Mandry (*29*).

In this outbreak, 9 of 18 persons regularly using water from a cistern, directly and heavily contaminated by carrier discharges, contracted typhoid fever. Of the nine regular users who escaped, one had had the disease previously and two others had had typhoid immunizations. Wilson (*16*) reports an outbreak at Lisnaskea Infirmary where 23 of 70 patients in the hospital contracted typhoid fever following the admission of a typhoid case to the hospital. This outbreak was apparently water-borne, the wells being subject to pollution by sewage of the institution. Isolations from three samples of well water (following the outbreak) gave estimated densities of 100, 87, and 30 typhoid bacteria per 100 ml.

Although the dividing line between the two chosen classifications of epidemics is not sharp, and no definite distinction can be made in many instances, the second group of outbreaks of typhoid fever might be considered as being due to "diffuse" infection. These epidemics would be characterized by low attack rates for typhoid fever, frequently preceded by widespread occurrence of gastro-intestinal disturbances. Such outbreaks have been so frequently recorded in the United States that one might expect such an outbreak in event of the failure of treatment processes, particularly chlorination, when treating a raw water grossly polluted with sewage. The general pattern of frequently occurring widespread gastro-enteritis, followed by a few cases of typhoid fever, is quite disturbing and the occurrence of such an oubreak in Great Britain at Kiddermaster led Sir George Newman (*30*) to state, "Nevertheless *B. typhosus* is still with us with all its potentialities for evil when conditions are favorable and the vehicle for its transference to the human being is forthcoming and set forth below is a brief account of a water-borne illness from which it is estimated more than 4,000 people in one town (of 29,000 population) suffered. There is strong circumstantial evidence, amounting to proof, that many of these imbibed the typhoid organism but only a very few contracted enteric fever" [9 cases of typhoid fever].

Bearing in mind the frequency of occurrence of this pattern of epidemic, widespread gastro-enteritis followed by a few cases of typhoid fever, and the usual concentrations of *E. typhosa* encountered in sewages and polluted waters as given in figure 1, one is forced to the conclusion that in general it seems unlikely that a single individual would, under such conditions, imbibe more than a single typhoid bacterium or at most only a very few. The theory is advanced, therefore, that a single typhoid bacterium is infective to a small percentage of the general population. An example of the nature of concentrations involved would, at this point, be interesting. Let it be assumed that a water plant is treating water with a typhosa/coliform ratio of 10 per million, corresponding roughly to that found by

Houston in the Thames and in London sewage. If filtered but unchlorinated water were distributed containing 500 coliforms per 100 ml., corresponding to positives for coliforms, in 5 of the 10-ml., 5 of the 1-ml., and 2 of 5 0.1-ml. portions, then the chance of an individual obtaining 1 *E. typhosa* in a daily portion of 1 liter of water would be about 1/20. Two *E. typhosa* from 1 liter of water would presumably be imbibed by 1 in 400 persons and 3 by 1 in 8,000 according to the laws of probability and assuming uniform distribution of bacteria. There would, of course, be little reason to expect that 3 typhosa would be much more infective than a single one. Yet such an occurrence, namely, the passage of 500 coliforms per 100 ml. of drinking water, in the absence of any knowledge concerning the concentration of *E. typhosa*, would, it is believed, give rise to outbreaks of gastro-enteritis followed by a few cases of typhoid fever in the majority of instances.

A question immediately arising is: What percentage of the general population would, on the average, develop a case of typhoid fever from the ingestion of a single *E. typhosa*? The definite answer to this question awaits a quantitatively controlled series of isolations of *E. typhosa* from samples of water supplies taken from the distribution system shortly after polluted water is by-passed or even during the early stages of the gastro-enteritis phase of the outbreak. The methods used by Houston (*11*, *12*) should be adequate if applied to sufficiently large quantities of water. This has probably never been accomplished in diffuse infections although there are records of isolations, following explosive outbreaks involving a high percentage of exposed individuals, such as the previously mentioned work by Wilson (*16*) and the isolations reported in a review of a paper by Klassen (*1*).

In an attempt to obtain an approximate estimate of the number of persons in a population group which might be expected to contract typhoid fever from ingestion of a single typhoid bacterium, according to the theory presented, a study was made of the data presented by Wolman and Gorman (*31*) in their book on water-borne typhoid fever outbreaks during the years 1920–1929. These authors classify outbreaks of water-borne gastro-enteritis and typhoid fever into 7 main groups and 27 subgroups according to the defect responsible for the condition causing the outbreak. Of these 27 subgroups, eliminating such causes as defects in the distribution system, underground supplies, and unknown supplies, the following classifications have been selected as those which would be expected to include largely "diffuse" infections:

A. Surface water supplies
   1. Contamination of brook or stream by pollution on watershed.
   2. Use of polluted river water—untreated.

3. Use of polluted lake water—untreated.
4. Contamination of spring, well, or infiltration gallery by pollution on watershed.
5. Contamination of spring, well, or infiltration gallery by flood waters.

B. Reservoirs or cistern storage
1. Seepage from sewer or surface into cracked cistern or reservoir.

C. Water purification
1. Inadequate control of filtration and allied treatment.
2. Inadequate chlorination—when this is the only treatment.
3. Interruption of chlorination—when this is the only treatment.

A total of 86 epidemics in these classifications was grouped and arranged in the order of increasing percentages of population contracting typhoid fever. A plot of this curve is shown in figure 6. It will be noted that the distribution of the outbreaks on the basis of the percentage of persons contracting the disease is such that for some distance the rise is approximately linear, departing from linearity noticeably when the attack rate is between 1 and 2 percent. If reasonably uniform distributions of concentrations of *E. typhosa* were present in these 86 outbreaks, one would expect a more or less sharp rise in the percentage of persons contracting the disease when exposed to more than a single bacterium. The percentage of persons contracting the disease, following ingestion of a single bacterium, based on the deviation from linearity in figure 6 would seem, therefore, to be in the neighborhood of 1 to 2 percent rather than, for example, 8 percent or more, or even some smaller figure such as 0.5 percent or less. This sort of reasoning is, of course, extremely crude, but it is valuable as a first approximation for estimating the expected cases in specific outbreaks.

Attempts have been made, therefore, to determine whether it is possible to account for certain epidemics by the presence of *E. typhosa* in typhosa/coliform ratios which have been reported in sewage, using 1.5 percent as the attack rate for typhoid fever following ingestion of a single bacterium. In only a few instances has it been possible to get sufficient data on "diffuse" infections to make even a rough estimate of the concentration of *E. typhosa* in the water supply responsible for the epidemic.

Santa Ana, Calif., 1924.—This outbreak was reported by Halliday and Beck (*32*) and as a news item in the Engineering News-Record (*33*). The outbreak involved some 300 cases of typhoid fever and 10,000 cases of gastro-enteritis in two waves. On December 28, 1923, due to a surcharged sewer system following a storm, there was pumped into one of the city reservoirs an estimated 49,000 gallons of 10 percent raw sewage (7 hours at 168,000 gallons per day), which was later pumped into the city mains. Dilution was such that no complaints were received of tastes and odors, thus indicating fairly good mixing

of the sewage and water. Using an estimated coliform content of 200,000 coliforms per ml. for a rather dilute sewage and a typhosa/coliform ratio of 20 per million for the sewage corresponding to an annual morbidity rate of 0.8 per 1,000 population (fig. 1), about 400 *E. typhosa* would be expected in each liter of 10-percent sewage or 74,000,000 typhosa in the 49,000 gallons. Of these about 0.1 percent would presumedly be ingested, or 74,000 *E. typhosa*. If these were uniformly mixed and passed out among the 30,000 population, the average number would be 2.5 per person, making no allowance for a probably rather low rate of natural decrease of *E. typhosa* in the reservoir and distribution system. Inasmuch as about one percent of the popula-

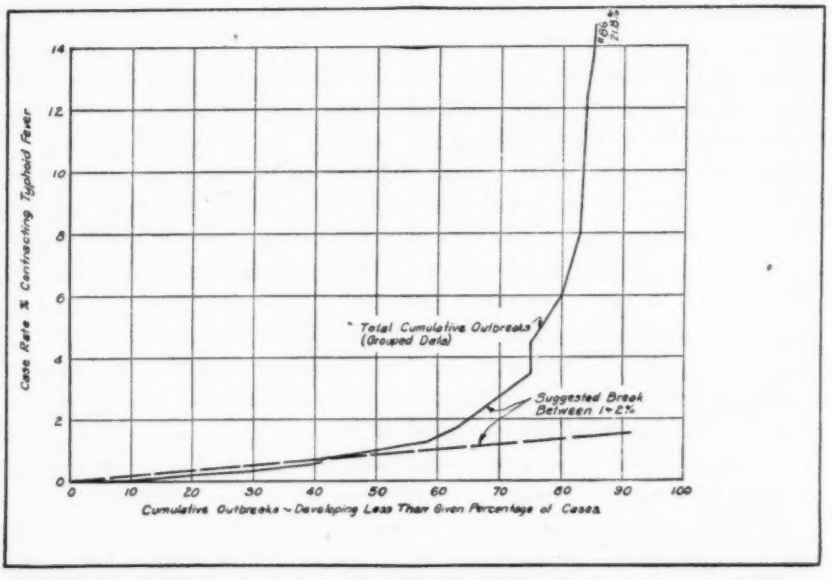

FIGURE 6.—Chart showing break in the linear relationship between cumulative numbers of typhoid fever outbreaks plotted against percentage of cases developing among the general population. Data from Wolman and Gorman (*31*).

tion contracted typhoid fever, it can be seen that the incidence of typhoid fever due to the *E. typhosa* in normal sewage is of the same magnitude as would be expected assuming that 1.5 percent of individuals exposed to a single bacterium would contract the disease. The inaccuracies in the base data must be borne in mind, of course.

Epidemic at Ponce, Puerto Rico (1938).—This epidemic was reported by Lopez (*34*) and involved 198 cases of water-borne typhoid fever in a population of 64,000. Records of the water plant indicate that about three to four of each five 10-ml. portions examined were positive for coliforms during the period July 1 to August 10, inclusive, giving an estimated density of about 10 coliforms per 100 ml. With a consumption of 1 liter per day, the average coliform intake during this 40-day period would be 4,000 per person. Typhoid fever cases had

averaged 50 per year for the preceding 2 years, or about 0.8 cases per 1,000 population, which from figure 1 would indicate an expected 20 *E. typhosa* per million coliforms in the polluted raw water. Using these assumptions, there would be 1 typhoid bacterium to 50,000 coliforms or 1 exposure to each 12.5 persons. If only 1.5 percent of those exposed to a single bacterium contracted the disease, it is necessary to assume a ratio of 1 *E. typhosa* to 20,000 coliforms to account for the reported cases. Such an assumption would, however, be in accord with known conditions, inasmuch as a single carrier can excrete tremendous numbers of typhoid bacteria. Furthermore, the assumption of a higher minimum infectious dose than a single bacterium for typhoid fever would, in this as in many instances, require a tremendous total infection of the water supply. In the particular epidemic under discussion it should be pointed out, however, that a more intense infection of shorter duration might easily have been the cause.

Detroit, Mich., February 1926.—This outbreak, involving 45,000 cases of gastro-enteritis and 8 cases of typhoid fever was reported by Wolman and Gorman (*31*). Data on the coliform content of the treated water were obtained by private communication. In this outbreak, the coliform content of the treated water at the filtration plant was listed as zero for all days of the month of February 1926, except the 25th and the 26th, when the average M. P. N. was 3.0 and 10.0 per 100 ml., respectively. If the mean intake of city water per person is estimated at about 0.5 liter for 2 days (in winter) with a concentration of 6.5 coliforms per 100 ml., and the 1925 mortality rate of 2.7 deaths per 100,000 be multiplied by 10 to get an average morbidity rate of 0.27 cases per 1,000 population, then from figure 1 the expected concentration of *E. typhosa* in the sewage which polluted the raw water and was presumably responsible for the epidemic would be 14 *E. typhosa* per million coliforms. From this the number of persons ingesting a single typhoid bacterium would be $\frac{32.5\times14}{1,000,000}\times1,300,000$ population=600. If 1.5 percent of those ingesting typhoid bacteria contracted the disease, 9 cases of typhoid fever would have developed, an expectancy only slightly greater and of the same order of magnitude as that which actually occurred.

PRESENT WATER SUPPLY PRACTICE AS RELATED TO THE THEORETICAL MINIMUM INFECTIOUS DOSE OF *E. TYPHOSA*

The question of occasional cases of typhoid fever from water supplies which meet present accepted standards of about 1 coliform per 100 ml. is quite an important problem. It will be remembered, however, that the estimated limit of purification previously considered

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for a constant typhosa/coliform ratio was about 99.9 percent purification from sewage or diluted sewage. Most raw waters for surface water supplies have already undergone natural purification of this order of magnitude or greater. To this must be added, from data published by Streeter (*36*, table 5–b), annual average purification plant removal for coliforms which he found to vary from 96.6 to 99.9995 for waters of the Great Lakes. Thus it will be seen that in normal water-plant operation a problem is presented of the fate of typhoid bacteria, relative to that of coliforms, in extremely low concentrations subjected to rather severe conditions.

While there is no widespread typhoid fever due to water supplies which meet present standards, as proved by present low typhoid rates, there is equally no assurance that an occasional case of waterborne typhoid fever does not develop under such conditions.

In fact, if the theory of single bacterium infection is valid, there are undoubtedly occasional cases of typhoid fever due to water supplies which meet approved standards. Such an event would probably only occur under conditions favorable to the passage of the typhoid bacterium and would probably require a high initial typhosa/coliform ratio in the raw water. Suggestions of such an occurrence are indicated by the Minneapolis typhoid fever epidemic of 1935 (*37*). The investigators of this outbreak could find no common source of infection other than water from the Fridley purification plant which had rarely been positive for coliform organisms in 10-ml. portions, although during the period of its presumed infectiveness low chlorine residuals were present and there were considerable numbers of lactose-fermenters which did not confirm as coliforms. The epidemic involved 214 cases scattered over a 3-month period. As part of the investigations following this outbreak, Heathman, Pierce, and Kabler (*38*) studied the comparative resistance of various strains of *E. typhosa* and coliforms to chlorine and chloramine. They found a considerable variation in the resistance of different strains of *E. typhosa* and coliforms and also found that certain freshly isolated strains of *E. typhosa* were more resistant than those which had been grown for some time upon artificial media. It is difficult to accept their implied suggestion of chlorine resistant pathogens in the Minneapolis outbreak, however, in view of their own findings of the variation in resistance of individual strains of both groups of organisms to chlorine and the obviously much larger number of strains of coliforms compared to those of *E. typhosa* which are normally present in sewage polluted waters. The exact effect of chlorine upon the large numbers of strains of the coliform group compared to the effect upon the few strains of *E. typhosa* present is problematical especially after decreases in bacteria have

continued to the range of potable waters. It is believed, however, that this ratio, $\frac{E.\ typhosa}{\text{coliforms}}$, would tend to be decreased in chlorinated waters.

It is interesting to note that Phelps (*39*) in his early experiments on the comparative resistance in aqueous suspension of *B. typhosus* and *B. coli* to calcium hypochlorite concluded that "the slight differences shown by the experiments on the two organisms may be attributed to experimental variations."

The averaged results of 12 sets of observations were as follows:

Time:	Percent removal	
	B. typhosus	*B. coli*
20 minutes	90.5	92.0
40 minutes	98.2	98.0
1 hour	99.45	99.53
2 hours	99.60	99.70
4 hours	99.92	99.96
18 hours	99.99+	99.99+

Average available chlorine was 5.0 p. p. m.

Consideration in this report of water-borne outbreaks due to pathogens other than *E. typhosa* has been limited owing to the lack of knowledge of the absolute infectiveness of such pathogens, the concentrations in which they occur, and their rates of decrease under natural conditions.

SUMMARY

A summary has been made of available quantitative data on the relative prevalence of coliforms and *E. typhosa* in sewage and sewage-polluted waters and presented as a ratio of *E. typhosa* per million coliforms at varying levels of typhoid fever morbidity in the community contributing such pollution. Evidence is presented that such a ratio could be expected to remain constant through bacterial reductions, due to natural purification processes approximating 99.9 percent. Data are also summarized regarding rates of decrease of coliforms and *E. typhosa* in three temperature ranges.

A theory is presented that the minimum infectious dose of *E. typhosa* in man is a single bacterium. Based on this theory, and the relative concentration of *E. typhosa* and coliforms usually present in the sewage from large populations, it is estimated that only a small percentage, possibly 1 or 2 percent of persons who ingest a single *E. typhosa*, develops typhoid fever.

The studies made herein of the available data in the literature emphasize the basic value of the coliform test as an indicator of the possible presence of pathogens, and indicate that a very real danger may exist when coliforms, in even moderately high concentrations, are present. The factor of safety provided by the ratio of a million or so coliforms present for each *E. typhosa* would, it is believed, take care of

usual fluctuations in the ratio of *E. typhosa* to coliforms provided the density of coliforms in ingested media be kept quite low or eliminated by methods which reduce the general bacterial population.

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THE TOXICITY OF LEAD AZIDE[1]

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The increased use of lead azide as a detonator in shells and the enormous expansion of the explosives industry in the war effort has stimulated interest among industrial hygienists with reference to possible harmful effects of exposure to lead azide or its intermediates in the process of manufacture. Apparently no investigation of the toxicity of lead azide itself has been made, although some investigation of hydrazoic acid has been reported (*1*).

Lead azide is a white crystalline substance having the formula $PB(N_3)_2$. It is very explosive, decomposes when warmed, and is sensitive to light. When exposed to sunlight it becomes covered with a dark brown film. Lead azide may be prepared by precipitation from an aqueous lead salt solution with hydrazoic acid or by a solution of a

[1] From the Division of Industrial Hygiene, National Institute of Health.

soluble azide. It is prepared commercially by precipitation from a solution of a soluble lead salt such as lead nitrate by the addition of a solution of sodium azide. A small quantity of dextrine is added to the solution in order to control crystal shape. If dextrine is not used, the lead azide crystals are needle-shaped, which not only are very sensitive but do not run freely in the charging machines at the later stages of manufacture (*2*). Sodium azide is prepared by the action of nitrous oxide on sodamide, $NaNH_2$. A basic lead azide can be prepared which is less sensitive to percussion or temperature than lead azide itself. When mixed with 30 percent water, lead azide is said to have the same sensitiveness as the dry material (*3*). According to Riegel (*4*), lead azide is about half as sensitive as mercury fulminate. It is said to have several advantages over mercury fulminate, $Hg(ONC)_2$, however. According to Stuart (*5*), it possesses a very considerably higher ignition point and is completely and permanently stable when exposed to temperatures of about 50° C. According to the same authority it is unaffected by other metals, shows a great superiority as an initiator of detonation, retains its brisance unimpaired under heavy pressure, and does not exhibit the phenomenon of becoming "dead pressed."

Hydrazoic acid, or azoimide, HN_3, in aqueous solution behaves as a strong acid dissolving such metals as zinc, iron, or magnesium with the evolution of hydrogen and the formation of the corresponding metallic salt. The acid solution has a penetrating, unpleasant odor, causes headache and eye irritation, and consequently requires care in handling.

Lead azide is coarsely crystalline in form and, owing to its explosive character, dust arising from it is kept to a minimum in industry. Exposure to the substance by inhalation in industry is consequently negligible. However, the possibility of inhalation of a certain amount of azoimide vapor occurs in the preparation of the lead compound and furthermore there is the possibility of other exposure to either the lead salt or more soluble azides in the course of preparation. For these reasons it is advisable to define more clearly the toxic factors associated with the industrial production of lead azide.

METALLIC AZIDES

Experiments were arranged in which three groups of ten white rats each received, by mouth, approximately 60 mg., 40 mg., or 20 mg. of lead azide per rat per day. A similar group of rats received 60 mg. of lead as lead carbonate per day. Finally two groups of rats of the same age as the preceding animals received the same basic diet and served as controls.

The deaths occurring in these various groups of lead azide rats were proportional to the amount of lead salt received, i. e., 100 percent of the rats given 60 mg. died within 9 weeks, 100 percent of the animals given 40 mg. died within 14 weeks, and 60 percent of the animals given 20 mg. were dead within 44 weeks. However, only 20 percent of the group of rats given lead carbonate were dead at the end of 44 weeks while 30 percent of the control animals had died within this period (fig. 1). The physical appearance and early death of those animals receiving the larger amounts of lead azide attested to the toxicity of the salt.

On the basis of this experiment alone it would appear that the

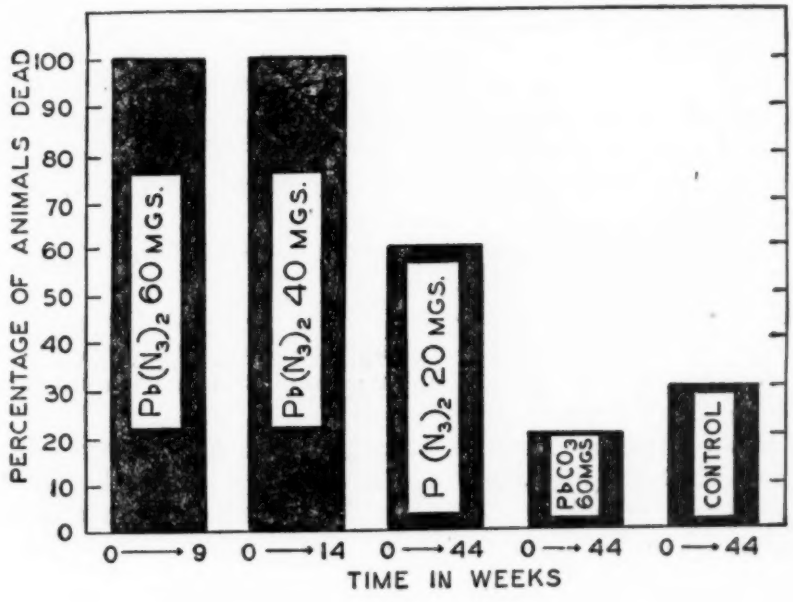

FIGURE 1.—The lethal action of ingested lead azide.

azoimide group rather than the lead is the more toxic molecular constituent.

The amounts of lead stored in the tissue of the rats of the various groups are indicated in table 1. It is apparent from these data that while lead was stored in all cases, no relation exists between storage at various levels of lead azide intake so far as this investigation is concerned. Those animals which received less lead azide per day lived longer and therefore eventually received a total amount of lead similar to or in excess of that received by the heavy dosage group. Therefore it was not surprising that the total amount stored was equal to or in excess of that stored by the animals which received the heavier dosage.

The concentration of lead in the livers of the animals receiving the higher dosage of lead azide was greater (0.266 mg./10 g. of liver) than is usual with lead-poisoned animals, but no striking differences were noted in the lead content of the kidney. The lead content of the bone

TABLE 1.—*The distribution of lead in the tissues of rats following the ingestion of lead azide*

Type of experiment	Average initial weight in grams	Average weight at death in grams	Percent mortality at end of 300 days	Average lead content of tissues mg. Pb/10 grams tissue		
				Liver	Kidney	Bone
Lead azide 20 mg./day	217	183	60	0.023	0.287	7.09
Lead azide 40 mg./day	295	150	100	.191	.480	4.87
Lead azide 60 mg./day	203	188	100	.266	.441	5.20
Lead carbonate 60 mg./day	158	213	20	.062	.498	11.28
Control I	236	211	30	.001	.001	.000
Control II	224	240	30	.001	.001	.001

was highest (11.28 mg/10 g. bone) in that group of animals which received 60 mg. of lead as carbonate for 10 months, and next highest (7.09 mg./10 g. bone) in the 20 mg./day lead azide group (fig. 1).

The amount of lead stored was therefore somewhat proportional to the total amount received and is not an index of toxicity. The earlier deaths of the animals receiving large doses of lead azide, on the other hand, indicate a toxicity inherent in that salt. As the preceding experiment indicated that the toxicity associated with lead azide was chiefly due to the azoimide anion, further study was made of hydrazoic acid and its corresponding sodium salt.

Sodium azide was added to the diet of a group of nine rats in an amount (26.7 mg. of sodium azide daily) equivalent to that fed the rats given 60 mg. of lead azide. The degree of mortality following the oral administration of this salt is shown in table 2.

TABLE 2.—*Effect of feeding sodium azide to nine rats equivalent in amount (26.7 mg.) to that of the rats fed 60 mg. lead azide*

Number of rats surviving	Average weight of rats in grams	Number of days on test	Percent mortality	Number of rats surviving	Average weight of rats in grams	Number of days on test	Percent mortality
9	224	0	0	5	170	26	44
9	207	7	0	4	168	30	55
9	193	14	0	3	165	33	67
8	185	17	11	2	159	38	78
7	181	20	22	1	151	39	88
6	174	25	33		150	39	100

It is evident from the above data that sodium azide is more acutely toxic than lead azide. The animals given sodium azide showed a 100 percent mortality in 39 days while the corresponding animals given lead azide attained 100 percent mortality only within 55 days.

Furthermore, a more pronounced drop in weight occurred with the

rats fed sodium azide. From an initial average weight of 224 gm. the weight dropped consistently to 150 gm. With the rats fed lead azide, on the other hand, the weight dropped from an initial value of 203 gm. to only 188 gm. It is apparent that the sodium salt is more rapidly fatal than the lead azide.

Further experiments with sodium azide in comparison with lead azide confirmed this finding. The intraperitoneal injection, subcutaneous injection, and oral administration of sodium azide and of lead azide indicated that weight for weight the former was the more toxic.

Some difficulty was encountered in securing and measuring the suspension of lead azide for injection owing to the rate at which it settled from suspension. However, sufficient data were secured to indicate that no more than 150 mg. per kg. of body weight could be injected intraperitoneally into rats without causing death. This would correspond to 33 mg. of sodium azide. In table 3 the physiological effect is indicated by various methods of administering sodium azide to rats in varying concentrations. The minimum lethal dose represented by the amount which caused 75 percent of the animals to die in 3 hours following injection lies between 35 and 38 mg.

TABLE 3.—*The effect of injecting sodium azide intraperitoneally and subcutaneously and the effect of oral administration of sodium azide in rats*

Number of rats	Dose in mg. per kg. of body weight	Equivalent of hydrazoic acid in mg.	Died in 3 hours	Percent dead in 3 hours
Intraperitoneal injection:				
4	25	16.5	0	0
11	30	19.8	1	10
12	33	21.8	8	66
15	35	23.1	10	66
5	36	23.8	4	80
5	37	24.4	5	100
Subcutaneous injection:				
5	33	21.8	0	0
9	35	23.1	4	45
8	38	25.1	8	100
Oral administration:				
3	40	26.4	0	0
2	42	27.8	1	50
2	44	29.1	1	50
8	45	29.8	5	62
3	46	30.4	3	100
3	48	31.7	3	100
3	60	39.7	3	100

The acute toxicity associated with lead azide and with sodium azide therefore should be evaluated in terms of their azoimide content.

Patch tests were made on 10 individuals with a 10-day interval between applications. No reaction whatsoever was observed in any case from either of these applications. Schwartz (*6*) has never found dermatitis from lead azide in industry.

HYDRAZOIC ACID

Although hydrazoic acid was prepared in several ways, a method based on Thiele's reaction was found to be especially useful. In this procedure 16.5 percent phosphoric acid solution was distilled with hydrazine sulfate and sodium nitrite. When smaller quantities were desired, hydrazoic acid was prepared by acidifying a solution of sodium azide, extracting the hydrazoic acid with ether, and evaporating the ether layer over distilled water. Where exposure to azoimide gas was desired, air was bubbled through an aqueous solution of acid displacing the azoimide.

Hydrazoic acid has a sickly penetrating odor and produces unpleasant after-effects when inhaled. The most marked effect noticeable is the eye irritation and severe headache following inhalation in low concentrations; in greater concentrations it causes death. The pure acid is a colorless liquid boiling at 37° C. and readily soluble in water or alcohol.

Since the pure acid is only of academic interest in the present instance, the properties of its solution in water are more pertinent to the present study. The aqueous solution smells strongly of azoimide. It precipitates azides from a number of metallic salt solutions and readily dissolves zinc, iron, magnesium, and aluminum with evolution of hydrogen and formation of the corresponding azide. The red color which hydrazoic acid gives with ferric salts was used as a means of evaluating concentrations of the acid where titration or gravimetric methods were inadequate.

A dilute aqueous solution of hydrazoic acid having a concentration of 1 percent was used in the following experiments. When added to a dilute solution of silver nitrate a white precipitate of silver azide formed which readily detonated when dry. Although dilute, the solution of hydrazoic acid smelled strongly and azoimide was readily given off when air was bubbled through the solution.

The hydrazoic acid for this purpose was prepared by acidifying an aqueous solution of sodium azide with sulfuric acid, extracting with ether, and evaporating the ethereal extract until only a trace of ether remained. The resulting aqueous solution of hydrazoic acid was diluted to various concentrations for use in the animal exposure experiments. The percentage composition of these various dilutions was determined by titration.

In order to determine whether toxic material was evolved from the aqueous solution of hydrazoic acid, air was bubbled into the solution, and animals exposed to this vapor.

Mice, rats, and guinea pigs were used as experimental animals. The results thus obtained are given in table 4.

TABLE 4.—*Effect of exposure of animals to vapor from various concentrations of hydrazoic acid solution*

Kind of animal	Number used	Volume of hydrazoic acid used (ml.)	Strength of hydrazoic acid solution—percent of HN_3 by weight	Time of lethal exposure in minutes
Mice	3	10	0.6	6
	3	10	.73	5
	3	5	.73	6
Rats	1	10	.6	28
	1	10	.6	31
	1	30	.6	7
	1	20	1.2	4
Guinea pigs	1	53	.6	80
	1	20	1.2	22

It is clearly evident from these results that azoimide can be readily displaced from aqueous solution by passing air through it and also that the azoimide vapor is decidedly toxic. Inhalation of the vapor caused a marked physiological response. This was noted after only a few moments of exposure. A degree of excitability was apparent first of all, followed by dyspnea, paralysis of the hind legs, convulsions, convulsive breathing, and finally death.

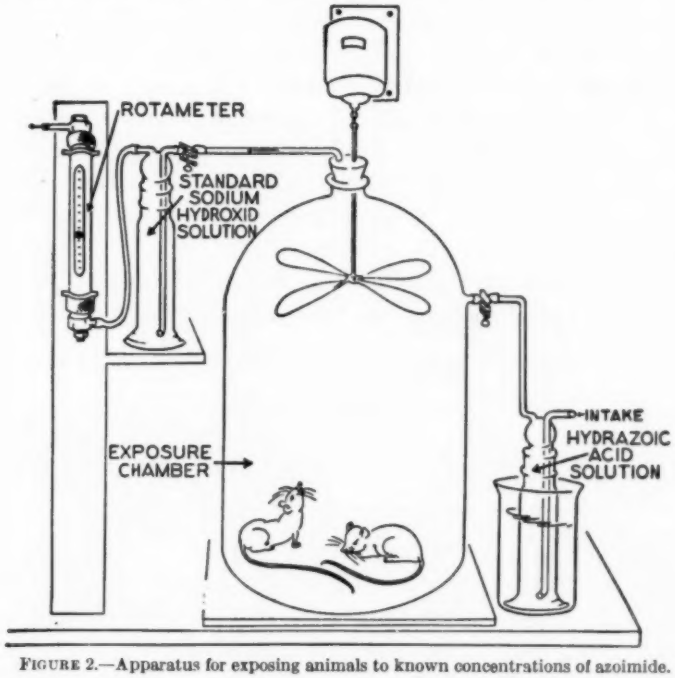

FIGURE 2.—Apparatus for exposing animals to known concentrations of azoimide.

Since an aqueous hydrazoic acid solution was found to give off hydrazoic acid gas in sufficient amount to prove fatal to animals, further experiments were made to determine the toxicity of azoimide gas at various concentrations. For this purpose the apparatus

shown in figure 2 was used. This consisted of a 33 liter bell jar having tubulature on the side two-thirds of the distance to the top. The animals to be exposed were placed upon a square glass plate, the bell jar lowered over them and made air tight at this junction with lubriseal. A fan served to mix the incoming gas rapidly and to maintain a uniform mixture of gases. Air drawn into the apparatus first passed through an aqueous solution of hydrazoic acid maintained at constant temperature in a water bath at 50° C. It then passed into the bell jar and any excess on leaving the bell jar was absorbed in a measured amount of standard sodium hydroxide solution in an absorption flask. The volume of air drawn into the apparatus was measured by means of a rotameter. A measured volume of hydrazoic acid, the concentration of which had been previously determined by titration, was placed in the bubbler and air passed through it until the concentration of gas in the bell jar was sufficiently high. The pinch clamps were then closed and the animals exposed to the given gas concentration for 1 hour. The air was kept in motion by the fan during the entire exposure. Both the solution in the hydrazoic acid bubbler and the sodium hydroxide solution of the absorption apparatus were titrated at the conclusion of the experiment. These various data enabled one to calculate the atmospheric gas content of azoimide in the bell jar. The animals were thus exposed to a known concentration of hydrazoic acid and observed for a period of 3 hours thereafter. A degree of excitability was apparent first of all, followed by dyspnea with flank breathing, lachrymation, salivation, and loss of muscular coordination of the extremities. These phenomena were followed by clonic convulsion, then tonic convulsion and death.

The data indicating the effect on animals following exposure to hydrazoic acid gas of various concentrations are given in table 5.

TABLE 5.—*Physiological response to various concentrations of hydrazoic acid gas (azoimide)*

Number of rats	Weight of rats in grams	Parts of azoimide per million parts of air	Number of deaths following 1 hour of exposure	Average percent mortality
2	245 215	849	0	
2	180 230	853	0	
2	240 190	853	0	
2	205 180	853	0	0
2	210 185	910	0	
2	215 190	910	0	
2	180 205	967	0	
2	210 245	1,024	1	37
2	240 235	1,024	2	

TABLE 5.—*Physiological response to various concentrations of hydrazoic acid gas (azoimide)*—Continued

Number of rats	Weight of rats in grams	Parts of azoimide per million parts of air	Number of deaths following 1 hour of exposure	Average percent mortality
2	218, 205	1,024	0	37
2	180, 185	1,024	0	
2	160, 240	1,081	1	75
2	208, 185	1,081	2	
2	260, 225	1,138	2	94
2	270, 235	1,138	2	
2	230, 280	1,138	2	
2	225, 200	1,138	2	
2	225, 215	1,138	1	
2	180, 185	1,138	2	
2	215, 190	1,138	2	
2	170, 220	1,138	2	
2	220, 235	1,162	2	100
2	195, 220	1,194	2	100
2	210, 235	1,194	2	
2	185, 200	1,251	2	100
2	245, 210	1,308	2	100
2	230, 205	1,308	2	
2	225, 240	1,365	2	100
2	188, 200	1,365	2	

These experiments indicate that a relatively sharply defined and consistent relationship exists between concentration and lethal action. Up to about 900 p. p. m., azoimide is not lethal when breathed by rats and guinea pigs for 1 hour. At a concentration beyond this point, however, its lethal effect is notable, and beyond about 1,160 p. p. m. its lethal action is invariable when it is breathed for 1 hour. It is invariably fatal in exposures of one-half to three-quarters of an hour to concentrations of about 1,300 p. p. m. When these values are plotted (fig. 3) the resulting graph tends to follow the sigmoid form characteristic for response of this type. On inspection of this curve it is apparent that a definitely pronounced effect becomes apparent above 1,100 p. p. m.

In comparison with certain other toxic gases it would appear that hydrazoic acid is lethal in concentrations approaching those of hydrogen sulfide or hydrogen cyanide, although its action is not so marked at low concentrations. On the other hand, the response with hydrazoic acid is sharply defined in the upper range of concentration (table 6).

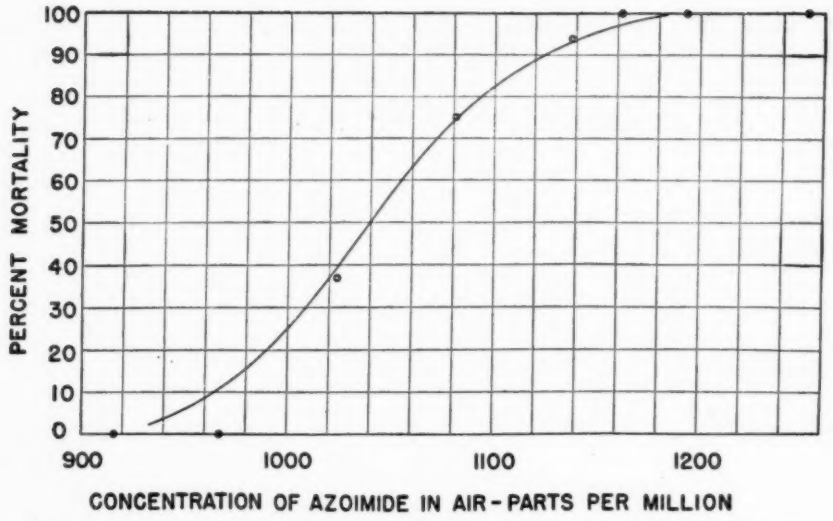

FIGURE 3.—The lethal action of azoimide at various concentrations following 1 hour of exposure.

TABLE 6.—*The physiological response of rats to azoimide of various concentrations in comparison with that of other noxious gases (7)*

Effect on the rat	Hydrazoic acid (p. p. m.)	Hydrogen sulfide (p. p. m.)	Hydrogen cyanide (p. p. m.)	Carbon monoxide (p. p. m.)
Not lethal when breathed for 1 hour	0–900			
Dangerous when inhaled for 1 hour	900–1, 100			
Fatal when breathed for 1½ hours			[1] 110 (a)	
First appearance of poisoning		150 (b)		
Fatal concentration at room temperature				1, 200 (c)
Assumes prone position after exposure of 9½ minutes			127 (d)	
Assumes prone position after exposure of 5 minutes			204 (e)	
Concentrations which are invariably fatal after exposure of 1 hour	1, 100–1, 300			
Concentration fatal in less than 1 hour	1, 300			
Recovery only if immediately removed following loss of consciousness		1. 500 (f)		
Concentration fatal following exposure of 10 minutes	2, 900			

[1] Letters after figures refer to reference (7).

In another experiment eight rats, in pairs, were exposed to higher concentrations and the time in minutes found necessary to kill each pair was noted. These values are given in table 7.

TABLE 7.—*Time of death following exposure of rats to high concentrations of azoimide*

Weight of rats in grams	Concentration of azoimide (p.p.m.)	Time of death in minutes	Weight of rats in grams	Concentration of azoimide (p.p.m.)	Time of death in minutes
180	1, 566	30	235	2, 080	16
200			200		
205	1, 872	19	200	2, 900	10
230			185		

The results obtained in this experiment show that these high concentrations are not only invariably fatal but that a concentration of the magnitude of 2,900 p.p.m. causes death when inhaled for as short a period as 10 minutes. This definitely places hydrazoic acid in the group of dangerous gases.

SUMMARY

An investigation of lead azide as an industrial hazard has indicated that the storage and distribution of lead in the tissues following the ingestion o this compound are in general similar to that of other lead salts. The acute toxic effect of this substance, however, is associated with the azoimide radical rather than with the lead.

Further evidence confirming this was obtained from observations of the effect of administering sodium azide intraperitoneally, subcutaneously, and orally in comparison with similar experiments with lead azide. The minimum lethal dose of sodium azide following injection lies between 35 and 38 mg. per kg. of body weight, while up to 150 mg. per kg. of body weight of lead azide could be injected intraperitoneally without causing death. This is equivalent in amount to 66 mg. of sodium azide.

The effect of exposure to hydrazoic acid gas by inhalation was determined at various concentrations and it has been shown to be invariably fatal to rats in amounts beyond 1160 p. p. m. when breathed for 1 hour. The results of this investigation indicate clearly that hydrazoic acid should be considered a dangerous gas.

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PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED APRIL 3, 1943

Summary

No significant change in health conditions in the United States is indicated by the reports of the important communicable diseases for the current week. With the exception of meningococcus meningitis and measles, the incidence of the 9 common communicable diseases included in the following tables is below or only slightly above the respective 5-year (1938–42) medians. A total of 26,183 cases of measles was reported, as compared with a median of 21,924.

A total of 595 cases of meningoccocus meningitis was reported, as compared with 572 for the preceding week and 614 for the next earlier week. As compared with the average numbers of cases reported in the past 3 weeks, increases are shown in the Middle Atlantic, East North Central, East South Central, and Pacific States. In the South Atlantic States a total of 106 cases was reported, as compared with 95 for the preceding week and a 3-week average of 120. Annual case rates per 100,000 estimated population for the first 13 weeks of the year, by geographic divisions, are as follows: New England, 34.2; Middle Atlantic, 17.8; East North Central, 7.6; West North Central, 10.9; South Atlantic, 26.0; East South Central, 19.4; West South Central, 10.9; Mountain, 17.4; and Pacific, 29.4. The rate for the United States is 17.5, as compared with 2.1 for the median of the past 5 years.

States reporting the largest numbers currently (preceding week's figures in parentheses) are as follows: New York, 68 (51); California, 58 (43); Mississippi, 43 (23); New Jersey, 39 (38); Pennsylvania, 38 (44); Virginia, 31 (33); Massachusetts, 23 (30); Maryland, 22 (17); Illinois, 21 (14); Kentucky, 20 (13); and Texas, 20 (20).

The total number of deaths recorded for the week in 89 large cities of the United States was 9,812, as compared with 9,858 for the preceding week and a 3-year average of 8,810. The cumulative figure for the first 13 weeks of the year is 130,970, as compared with 119,905 in the same period of last year.

Telegraphic morbidity reports from State health officers for the week ended April 3, 1943, and comparison with corresponding week of 1942 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended—		Median	Week ended—		Median	Week ended—		Median	Week ended—		Median
	Apr. 3, 1943	Apr. 4, 1942	1938–42	Apr. 3, 1943	Apr. 4, 1942	1938–42	Apr. 3, 1943	Apr. 4, 1942	1938–42	Apr. 3, 1942	Apr. 4, 1942	1938–42
NEW ENG.												
Maine	0	1	1			4	8	173	164	8	4	1
New Hampshire	0	0	0	5	6		60	8	46	1	0	0
Vermont	0	0	0				470	70	43	1	0	0
Massachusetts	3	5	2				1,665	1,085	787	23	7	2
Rhode Island	1	0	0	1			11	267	9	17	0	0
Connecticut	1	1	1	5		6	455	365	134	9	2	1
MID. ATL.												
New York	17	29	22	[1] 19	[1] 15	[1] 15	2,826	622	1,467	68	30	4
New Jersey	6	3	6	9	4	11	1,653	379	461	39	5	1
Pennsylvania	7	11	25	5			2,394	1,081	1,081	38	5	5
E. NO. CEN.												
Ohio	13	9	9	7	23	16	1,227	354	354	7	1	2
Indiana	3	6	6	44	23	27	761	125	125	9	0	1
Illinois	22	8	34	11	23	33	1,378	527	527	21	1	1
Michigan [2]	5	2	11	4	2	3	1,301	202	393	18	2	1
Wisconsin	1	4	0	36	55	202	1,563	870	870	12	1	1
W. NO. CEN.												
Minnesota	3	0	1			3	126	693	214	4	1	0
Iowa	5	1	3		5	14	341	267	267	2	0	0
Missouri	4	4	4	8	1	9	369	157	146	12	0	0
North Dakota	0	0	1	3		5	56	64	44	0	0	0
South Dakota	10	0	1			1	178	6	6	0	0	0
Nebraska	0	5	4	29	55		196	190	80	0	0	0
Kansas	7	1	4	3	12	12	629	646	580	4	2	1
SO. ATL.												
Delaware	0	0	0				95	3	3	2	0	0
Maryland [2]	16	1	3	8	5	41	140	780	393	22	5	1
Dist. of Col.	0	1	2	1	3	2	75	91	91	5	2	0
Virginia	3	3	14	556	311	441	621	217	421	31	3	1
West Virginia	1	6	9	119	22	36	90	209	209	4	3	3
North Carolina	8	8	12	71	26	37	93	1,090	1,090	18	2	1
South Carolina	3	7	5	473	605	605	175	347	347	15	2	1
Georgia	4	5	7	48	45	90	264	263	263	7	4	2
Florida	2	4	6	5	1	13	69	260	260	2	1	1
E. SO. CEN.												
Kentucky	3	7	7	7	9	30	543	111	111	20	4	4
Tennessee	3	2	6	74	44	153	540	129	129	18	1	2
Alabama	7	8	8	324	328	328	320	257	257	9	0	3
Mississippi [2]	2	7	6							43	1	1
W. SO. CEN.												
Arkansas	4	2	6	62	201	201	157	320	320	1	0	0
Louisiana	3	3	7	8	3	11	240	292	69	7	0	1
Oklahoma	1	4	7	78	141	197	107	255	111	1	0	1
Texas	29	39	26	1,129	1,113	1,154	1,297	2,139	789	20	7	2
MOUNTAIN												
Montana	2	0	0	52	5	10	374	150	44	0	0	0
Idaho	0	1	1				27	60	39	0	0	0
Wyoming	1	0	1	26	104	1	213	77	77	1	0	0
Colorado	14	6	7	31	49	30	720	254	272	4	1	0
New Mexico	0	0	2		3	3	12	133	110	0	0	0
Arizona	1	0	2	98	151	137	31	206	104	0	0	0
Utah [2]	0	0	0	9	7	13	252	235	235	1	0	0
Nevada	0	0		3			21	9		0	0	
PACIFIC												
Washington	7	0	1	2	1	1	775	286	286	6	3	1
Oregon	1	2	2	22	24	28	453	130	130	7	1	1
California	19	17	17	70	220	220	812	5,470	686	58	9	2
Total	242	223	311	3,465	3,645	4,087	26,183	21,924	21,924	595	110	57
13 weeks	3,679	4,037	4,942	57,434	61,452	113,646	210,408	204,951	204,951	5,826	952	682

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended April 3, 1943, and comparison with corresponding week of 1942 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended—		Median 1938–42	Week ended—		Median 1938–42	Week ended—		Median 1938–42	Week ended—		Median 1938–42
	Apr. 3, 1943	Apr. 4, 1942		Apr. 3, 1943	Apr. 4, 1942		Apr. 3, 1943	Apr. 4, 1942		Apr. 3, 1943	Apr. 4, 1942	
NEW ENG.												
Maine	0	0	0	13	13	13	0	0	0	1	0	0
New Hampshire	0	0	0	10	14	7	0	0	0	0	0	0
Vermont	0	0	0	10	11	15	0	0	0	0	0	0
Massachusetts	0	0	0	568	363	171	0	0	0	2	2	0
Rhode Island	0	0	0	17	19	17	0	0	0	0	1	0
Connecticut	0	0	0	93	30	86	0	0	0	0	0	0
MID. ATL.												
New York	0	2	1	559	498	748	0	0	0	4	6	6
New Jersey	0	0	0	204	117	181	0	0	0	1	1	1
Pennsylvania	2	0	0	350	494	461	0	0	0	0	1	7
E. NO. CEN.												
Ohio	0	2	1	314	414	414	1	0	3	2	0	2
Indiana	2	0	0	154	125	190	2	0	5	0	0	1
Illinois	0	0	1	271	238	512	1	1	7	6	1	3
Michigan [2]	0	1	1	119	219	396	0	0	0	3	0	2
Wisconsin	0	0	0	339	175	175	0	0	4	0	2	0
W. NO. CEN.												
Minnesota	0	0	0	33	89	89	0	0	3	1	0	0
Iowa	0	0	0	59	55	75	1	2	23	0	0	0
Missouri	0	0	0	80	37	40	0	3	17	1	0	1
North Dakota	0	0	0	5	21	15	0	0	3	0	0	0
South Dakota	0	0	0	11	42	13	0	0	1	0	0	0
Nebraska	0	0	0	29	60	44	0	0	0	0	0	0
Kansas	0	0	0	74	105	105	1	0	1	0	0	0
SO. ATL.												
Delaware	0	0	0	9	25	11	0	0	0	0	0	0
Maryland [3]	0	0	0	146	79	49	0	0	0	5	2	2
Dist. of Col	1	0	0	20	8	16	0	0	0	0	1	0
Virginia	0	0	0	43	18	32	0	0	0	2	0	3
West Virginia	0	0	0	21	39	42	0	0	0	0	4	4
North Carolina	0	0	0	32	27	32	0	0	0	0	1	2
South Carolina	0	1	0	5	2	4	1	0	0	1	6	3
Georgia	0	0	0	15	9	13	9	0	0	1	2	2
Florida	0	0	1	7	8	8	0	0	0	1	2	4
E. SO. CEN.												
Kentucky	3	0	0	51	71	96	0	0	0	0	1	2
Tennessee	0	0	0	45	48	67	0	5	3	3	2	2
Alabama	0	0	0	43	11	11	0	1	1	2	1	3
Mississippi [3]	1	2	2	10	9	7	1	0	0	1	3	2
W. SO. CEN.												
Arkansas	0	0	0	5	5	5	1	2	2	1	0	1
Louisiana	0	0	0	13	4	7	0	2	1	2	5	6
Oklahoma	0	0	0	17	15	18	0	1	3	1	2	1
Texas	0	0	0	162	60	60	13	18	18	3	7	5
MOUNTAIN												
Montana	0	1	1	16	32	29	0	0	0	0	0	0
Idaho	0	0	0	7	2	8	0	0	0	0	0	0
Wyoming	0	0	0	58	19	17	0	0	0	0	0	0
Colorado	0	1	0	39	37	37	0	0	7	0	1	0
New Mexico	0	0	0	3	4	11	0	0	0	4	1	1
Arizona	[3] 4	0	0	19	4	5	0	0	0	0	1	1
Utah [3]	1	0	0	49	24	23	0	0	0	0	0	0
Nevada	0	0	-----	1	2	-----	0	0	-----	0	0	-----
PACIFIC												
Washington	1	0	0	40	24	24	0	0	1	4	0	0
Oregon	0	0	0	12	12	18	0	1	1	1	0	0
California	5	0	0	136	92	149	0	0	2	1	3	4
Total	[3] 20	10	19	4,336	3,829	5,064	31	36	72	54	59	86
13 weeks	340	300	300	51,038	52,173	61,523	350	302	954	692	966	1,002

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended April 3, 1943, and comparison with corresponding week of 1942 and 5-year median—Con.

Division and State	Whooping cough			Week ended Apr. 3, 1943								
	Week ended—		Median 1938–42	Anthrax	Dysentery			Encephalitis, infectious	Leprosy	Rocky Mt. spotted fever	Tularemia	Typhus fever
	Apr. 3, 1943	Apr. 4, 1942			Amebic	Bacillary	Unspecified					
NEW ENG.												
Maine	60	19	33	0	0	0	0	0	0	0	0	0
New Hampshire	23	15	9	0	0	0	0	0	0	0	0	0
Vermont	27	35	34	0	0	0	0	0	0	0	0	0
Massachusetts	179	196	196	0	0	0	0	0	0	0	0	0
Rhode Island	43	43	36	0	0	0	0	0	0	0	0	0
Connecticut	51	83	67	0	0	1	0	0	0	0	0	0
MID. ATL.												
New York	405	500	393	0	21	23	0	2	0	0	0	0
New Jersey	205	180	180	0	0	0	0	1	0	1	0	0
Pennsylvania	311	180	349	1	0	0	0	0	0	0	0	0
E. NO. CEN.												
Ohio	181	157	209	0	0	0	0	1	0	0	0	0
Indiana	83	27	30	0	1	0	0	0	0	0	0	0
Illinois	111	157	118	0	4	1	0	2	0	0	4	0
Michigan [3]	253	131	174	0	0	3	0	0	0	0	0	0
Wisconsin	190	146	146	0	0	0	0	0	0	0	1	0
W. NO. CEN.												
Minnesota	78	23	29	0	1	0	0	0	0	0	0	0
Iowa	18	27	27	0	1	0	0	1	0	0	0	0
Missouri	8	2	12	0	0	0	0	1	0	0	0	0
North Dakota	16	1	26	0	0	0	0	0	0	0	0	0
South Dakota	5	0	2	0	0	0	0	0	0	0	0	0
Nebraska	8	3	9	0	0	0	0	0	0	0	0	0
Kansas	109	49	49	0	0	0	0	1	0	0	1	0
SO. ATL.												
Delaware	6	8	7	0	0	0	0	0	0	0	0	0
Maryland [2]	109	39	80	0	0	0	1	1	0	0	1	0
Dist. of Col.	42	15	14	0	0	0	0	0	0	0	0	0
Virginia	85	53	53	0	0	0	24	0	0	0	0	0
West Virginia	106	16	49	0	0	0	0	0	0	0	0	0
North Carolina	179	156	286	0	2	0	0	0	0	0	0	3
South Carolina	32	96	96	0	0	2	0	0	0	0	1	1
Georgia	42	29	29	0	2	0	0	0	0	0	8	9
Florida	30	23	23	0	0	0	0	0	0	0	0	12
E. SO. CEN.												
Kentucky	49	101	74	0	0	0	0	1	0	0	0	0
Tennessee	111	23	36	0	2	0	0	0	0	0	2	1
Alabama	52	51	51	0	0	0	0	0	0	0	0	2
Mississippi [2]	----	----	----	0	0	0	0	0	0	0	1	0
W. SO. CEN.												
Arkansas	26	7	18	0	0	7	0	0	0	0	1	0
Louisiana	10	24	23	0	0	0	0	0	0	0	0	0
Oklahoma	33	9	9	0	0	0	0	0	0	0	0	0
Texas	545	181	243	0	24	99	0	1	0	0	6	12
MOUNTAIN												
Montana	16	26	9	0	0	0	0	0	0	0	0	0
Idaho	5	4	5	0	0	0	0	0	0	0	0	0
Wyoming	2	3	3	0	0	0	0	1	0	0	0	0
Colorado	11	55	55	0	0	1	0	0	0	0	0	0
New Mexico	13	36	31	0	0	0	0	0	0	0	0	0
Arizona	29	45	37	0	0	0	4	0	0	0	0	0
Utah [3]	51	30	47	0	0	0	0	0	0	0	0	0
Nevada	0	8	----	0	0	0	0	0	0	0	0	0
PACIFIC												
Washington	30	90	79	0	0	0	0	0	0	0	0	0
Oregon	27	29	17	0	0	0	0	0	0	0	0	0
California	394	283	283	0	1	3	0	1	0	0	0	0
Total	4,399	3,414	4,110	1	59	140	29	14	0	1	26	40
13 weeks	51,424	50,708	54,013	20	383	2,602	546	141	5	4	231	637

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Delayed report of one February case in Arizona included.

WEEKLY REPORTS FROM CITIES

City reports for week ended March 20, 1943

This table lists the reports from 88 cities of more than 10,000 population distributed throughout the United States, and represents a cross section of the current urban incidence of the diseases included in the table.

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Atlanta, Ga	0	0	22	1	14	1	5	0	0	0	0	2
Baltimore, Md	5	0	2	4	37	18	27	0	67	0	0	82
Barre, Vt	0	0	------	0	1	0	0	0	0	0	0	0
Billings, Mont	1	0	------	0	0	0	0	0	0	0	0	0
Birmingham, Ala	0	0	7	3	1	1	10	0	3	0	0	1
Boise, Idaho	0	0	------	0	1	0	0	0	1	0	0	0
Boston, Mass	0	0	------	1	186	7	7	0	180	0	0	52
Bridgeport, Conn	0	0	1	1	3	0	2	0	3	0	0	1
Brunswick, Ga	0	0	------	0	2	0	2	0	0	0	0	0
Buffalo, N. Y	0	0	------	1	132	1	4	0	11	0	0	19
Camden, N. J	1	0	1	0	0	0	7	0	3	0	0	0
Charleston, S. C	1	0	65	0	0	0	2	0	1	0	0	0
Charleston, W. Va	0	0	2	0	1	0	0	0	0	0	0	1
Chicago, Ill	7	0	4	1	594	1	29	0	79	0	1	50
Cincinnati, Ohio	0	0	1	0	73	2	6	0	41	0	0	2
Cleveland, Ohio	1	0	7	1	17	1	12	0	56	0	0	56
Columbus, Ohio	0	0	2	2	11	0	6	0	16	0	1	4
Concord, N. H	0	0	------	0	0	0	0	0	0	0	0	0
Cumberland, Md	0	0	------	0	0	0	0	0	1	0	0	0
Dallas, Tex	0	0	1	1	9	0	4	0	7	0	0	11
Denver, Colo	5	0	19	0	492	0	11	0	17	0	0	6
Detroit, Mich	2	0	10	23	295	9	3	0	41	0	1	85
Duluth, Minn	0	0	------	0	2	0	3	0	7	0	0	3
Fall River, Mass	0	0	------	0	14	0	0	0	2	0	0	29
Fargo, N. Dak	0	0	------	0	4	0	0	0	0	0	0	0
Flint, Mich	0	0	------	0	10	0	9	0	8	0	0	0
Fort Wayne, Ind	0	0	------	0	1	0	4	0	11	0	0	0
Frederick, Md	0	0	------	0	5	0	0	0	0	0	0	0
Galveston, Tex	0	0	------	0	1	0	1	0	1	0	0	0
Grand Rapids, Mich	0	0	1	0	8	0	0	0	7	0	0	10
Great Falls, Mont	0	0	------	0	42	0	0	0	3	0	0	6
Hartford, Conn	0	0	------	0	37	0	8	0	5	0	0	9
Helena, Mont	0	0	------	0	80	0	0	0	0	0	0	1
Houston, Tex	2	0	------	1	7	0	9	2	2	0	0	3
Indianapolis, Ind	0	0	------	3	152	0	8	0	22	0	0	37
Kansas City, Mo	2	0	------	0	93	3	5	0	69	0	0	6
Kenosha, Wis	0	0	------	0	2	0	1	0	1	0	0	0
Little Rock, Ark	1	0	1	0	5	1	3	0	2	0	0	2
Los Angeles, Calif	4	0	17	2	124	4	8	1	36	0	0	33
Lynchburg, Va	0	0	------	0	0	1	2	0	0	0	0	4
Memphis, Tenn	1	0	1	3	81	2	7	0	12	0	0	26
Milwaukee, Wis	0	0	------	0	376	2	2	0	166	0	0	31
Minneapolis, Minn	3	0	------	0	30	2	5	0	18	0	0	12
Missoula, Mont	0	0	------	0	14	0	1	0	0	0	0	0
Mobile, Ala	0	0	2	2	1	0	5	0	2	0	0	3
Nashville, Tenn	0	0	------	1	63	0	5	0	2	0	0	4
Newark, N. J	0	0	4	0	148	2	5	0	11	0	0	15
New Haven, Conn	0	0	------	0	2	0	0	0	2	0	0	2
New Orleans, La	0	0	10	1	60	4	7	0	8	0	0	1
New York, N. Y	17	2	8	6	460	47	103	1	406	0	4	89
Omaha, Nebr	0	0	------	0	9	0	6	0	9	0	0	4
Philadelphia, Pa	0	0	------	1	928	12	42	0	113	0	0	88
Pittsburgh, Pa	0	0	7	4	13	2	18	0	9	0	1	40
Portland, Maine	0	0	------	0	0	3	4	0	1	0	0	14
Providence, R. I	0	0	17	0	2	5	5	0	3	0	0	29
Pueblo, Colo	0	0	------	0	4	0	1	0	1	0	0	13
Racine, Wis	0	0	------	0	13	0	0	0	30	0	0	5
Reading, Pa	0	0	------	0	197	1	1	0	4	0	0	7
Richmond, Va	0	0	------	1	7	5	4	0	4	0	0	0

City reports for week ended March 20, 1943—Continued

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Roanoke, Va	0	0	------	0	0	0	2	0	0	0	0	0
Rochester, N. Y	0	0	------	0	30	1	1	0	10	0	0	23
Sacramento, Calif	5	0	1	0	14	3	4	0	7	0	1	4
Saint Joseph, Mo	0	0	------	0	2	0	5	0	2	0	0	0
Saint Louis, Mo	0	0	8	2	60	14	9	0	17	0	0	10
Saint Paul, Minn	0	0	------	0	12	1	3	0	7	0	0	34
Salt Lake City, Utah	1	0	------	0	208	2	0	0	14	0	0	17
San Antonio, Tex	2	0	1	1	8	0	7	0	0	0	0	1
San Francisco, Calif	2	0	------	0	74	2	16	0	21	0	0	20
Savannah, Ga	0	0	27	6	4	0	3	0	0	0	0	0
Seattle, Wash	0	0	------	1	97	2	5	0	4	0	0	9
Shreveport, La	0	0	------	0	0	0	4	0	1	0	0	0
South Bend, Ind	0	0	------	0	15	0	0	0	3	0	0	1
Spokane, Wash	0	0	1	1	152	1	8	0	0	0	0	8
Springfield, Ill	0	0	------	0	8	0	2	0	4	0	0	8
Springfield, Mass	0	0	------	0	11	1	2	0	82	0	0	0
Superior, Wis	0	0	------	0	3	0	0	0	2	0	0	6
Syracuse, N. Y	0	0	------	0	29	0	4	0	23	0	0	29
Tacoma, Wash	0	0	------	0	23	0	3	0	1	0	0	0
Tampa, Fla	0	0	------	0	4	0	3	0	0	0	0	2
Terre Haute, Ind	0	0	------	0	11	0	1	0	0	0	0	4
Topeka, Kans	0	0	------	0	189	0	0	0	1	0	0	3
Trenton, N. J	0	0	1	0	69	0	3	0	9	0	0	2
Washington, D. C	1	0	4	2	100	3	9	0	16	0	0	26
Wheeling, W. Va	0	0	------	1	5	0	1	0	1	0	0	2
Wichita, Kans	0	0	------	0	40	0	0	0	1	0	0	8
Wilmington, Del	2	0	------	1	23	0	6	0	2	0	0	1
Winston-Salem, N. C	0	0	------	0	0	0	1	0	0	0	0	31
Worcester, Mass	0	0	------	0	381	1	5	0	13	0	0	4
Total	66	2	254	79	6,436	168	527	4	1,745	0	9	1,151
Corresponding week 1942	78	2	167	45	5,234	49	535	4	1,652	1	13	1,132
Average, 1938–42	92	------	459	[1] 59	[2]4,762	------	[1] 516	------	1,661	15	21	1,095

Anthrax.—Cases: Camden, 1.
Dysentery, amebic.—Cases: Boston, 1; New York, 14.
Dysentery, bacillary.—Cases: Buffalo, 1; Los Angeles, 3; New York, 1; Philadelphia, 8.
Dysentery, unspecified.—Cases: San Antonio, 3.
Tularemia.—Cases: Nashville, 1; New Orleans, 1.
Typhus fever.—Cases: Houston, 1; Savannah, 2.

[1] 3-year average, 1940–42.
[2] 5-year median.

DEATHS DURING WEEK ENDED MARCH 27, 1943

[From the Weekly Mortality Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Mar. 27, 1943	Corresponding week, 1942
Data for 89 large cities of the United States:		
Total deaths	9,858	9,040
Average for 3 prior years	9,001	
Total deaths, first 12 weeks of year	121,158	111,297
Deaths under 1 year of age	692	632
Average for 3 prior years	571	
Deaths under 1 year of age, first 12 weeks of year	8,588	6,870
Data from industrial insurance companies:		
Policies in force	65,462,918	65,017,199
Number of death claims	13,135	13,181
Death claims per 1,000 policies in force, annual rate	10.5	10.6
Death claims per 1,000 policies, first 12 weeks of year, annual rate	10.7	10.3

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended March 6, 1943.—During the week ended March 6, 1943, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Chickenpox	1	20		194	242	20	19	13	69	578
Diphtheria	2	14	4	21	5	5	1		1	53
Dysentery (amebic)						1				1
Dysentery (bacillary)				10						10
German measles		4		5	28	1	1	4	6	49
Influenza		4	9		173	11			44	241
Measles		21	2	140	270	51	197	21	69	771
Meningitis, meningococcus	1	1		3	3			1	2	11
Mumps	8	167	13	67	1,216	141	100	137	144	1,993
Scarlet fever		13	9	100	127	22	20	49	20	360
Tuberculosis (all forms)	2	2	7	100	49	15	6	3	17	201
Typhoid and paratyphoid fever				15						15
Whooping cough		1		53	103	36	6	15	12	226

NEW CALEDONIA

Notifiable diseases—Year 1942.—During the year 1942, certain notifiable diseases were reported on the island of New Caledonia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Cerebrospinal meningitis	27	6	Poliomyelitis	1	
Diphtheria	3		Tuberculosis	16	11
Plague (human)	2	1	Typhoid fever	9	5

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—Except in cases of unusual prevalence, only those places are included which had not previously reported any of the above-mentioned diseases, except yellow fever, during the current year. All reports of yellow fever are published currently.

A cumulative table showing the reported prevalence of these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday in each month.

(Few reports are available from the invaded countries of Europe and other nations in war zones.)

Typhus Fever

Bulgaria.—For the period January 14 to February 3, 1943, 136 cases of typhus fever were reported in Bulgaria.

Hungary.—For the week ended March 13, 1943, 56 cases of typhus fever were reported in Hungary.

Rumania.—For the period March 8–15, 1943, 497 cases of typhus fever, including 34 in Bucharest, were reported in Rumania.

Union of South Africa.—During the month of December 1942, 224 cases of typhus fever were reported in Union of South Africa.